

09/593,828

=> d his

(FILE 'HOME' ENTERED AT 11:06:03 ON 20 JUL 2004)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS, LIFESCI' ENTERED AT 11:06:28 ON 20 JUL 2004

L1 2714 S GLYCOSYL(A)TRANSFERASE?
L2 1105 S "GST-ALPHA" OR GST4(W)ALPHA
L3 3819 S L1 OR L2
L4 1242 S HUMAN AND L3
L5 50 S HUMAN(W)L3
L6 83 S HUMAN(2W)L3
L7 6615667 S CLON? OR EXPRESS? OR RECOMBINANT
L8 1242 S L4 OR L5 OR L6
L9 700 S L7 AND L8
L10 14569 S L(W)SELECTIN OR "L-SELECTIN"
L11 0 S L9 AND L10
L12 55768 S SELECTIN
L13 3 S L9 AND L12
L14 3 DUP REM L13 (0 DUPLICATES REMOVED)
L15 63891 S "GLYCAM-1" OR "CD34" OR "MADCAM-1" OR "SGP200" OR "PODOCALYXI
L16 9 S L9 AND L15
L17 4 DUP REM L16 (5 DUPLICATES REMOVED)
L18 33 S L5 AND L7
L19 12 DUP REM L18 (21 DUPLICATES REMOVED)
E ROSEN S/AU
L20 2426 S E3
L21 0 S L3 AND L20
L22 23 S GLYCOSYL (2W)SULFOTRANSFERASE?
L23 0 S L20 AND L22
L24 6 S HUMAN (2W)L22
L25 6 DUP REM L24 (0 DUPLICATES REMOVED)

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NEWS 3 May 12 EXTEND option available in structure searching
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SDIs in CPlus
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FILE 'HOME' ENTERED AT 11:06:03 ON 20 JUL 2004

=> file medline embase biosis biotechds scisearch hcaplus ntis lifesci
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FULL ESTIMATED COST 0.21 0.21

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=> s g;ycosyl(a)transferase?
<-----User Break----->

SEARCH ENDED BY USER

=> s glycosyl(a)transferase?
L1 2714 GLYCOSYL(A) TRANSFERASE?

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L5 50 HUMAN(W) L3

=> s human(2w)l3
L6 83 HUMAN(2W) L3

=> s clon? or express? or recombinant
5 FILES SEARCHED...
L7 6615667 CLON? OR EXPRESS? OR RECOMBINANT

=> s l4 ot l5 ot l6
MISSING OPERATOR L4 OT
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s l4 or l5 or l6
L8 1242 L4 OR L5 OR L6

=> s l7 and l8
L9 700 L7 AND L8

=> s L(W)selectin or "L-selectin"
L10 14569 L(W) SELECTIN OR "L-SELECTIN"

=> s l9 and l10
L11 0 L9 AND L10

=> s selectin
L12 55768 SELECTIN

=> s l9 and l12
L13 3 L9 AND L12

=> dup rem l13
PROCESSING COMPLETED FOR L13
L14 3 DUP REM L13 (0 DUPLICATES REMOVED)

=> d 1-3 ibib ab

L14 ANSWER 1 OF 3 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
ACCESSION NUMBER: 2002:473287 SCISEARCH
THE GENUINE ARTICLE: 558WH
TITLE: Plasmid vectors with a 5 '-hybrid intron facilitate
high-level glycoprotein **expression** in CHO-cells
AUTHOR: Melcher R (Reprint); Grosch H W; Hasilik A
CORPORATE SOURCE: Univ Wurzburg, Dept Med, Gastrolabor Bau 4, Joseph
Schneider Str 2, D-97080 Wurzburg, Germany (Reprint); Univ
Wurzburg, Dept Med, D-97080 Wurzburg, Germany; Univ
Marburg, Inst Physiol Chem, D-35033 Marburg, Germany
COUNTRY OF AUTHOR: Germany
SOURCE: BIOCHIMICA ET BIOPHYSICA ACTA-GENE STRUCTURE AND
EXPRESSION, (3 MAY 2002) Vol. 1575, No. 1-3, pp. 49-53.
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE
AMSTERDAM, NETHERLANDS.
ISSN: 0167-4781.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 23

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB For biosynthesis of **recombinant** glycoproteins with specified
carbohydrate structures various Chinese hamster ovary (CHO) cell lines are
available that **express** different sets of **glycosyl**
transferases. To examine various forms of glycosylated lysozyme we
prepared a vector that directs the synthesis of the **recombinant**
glycoprotein at a high rate. We compared vectors with varied promoter and
5'-untranslated regions. The **expression** of cDNA of a
glycosylated mutant lysozyme was examined under a control of the SV40
early and cytomegalovirus (CMV) promoters alone and in combination with a
tripartite leader and a hybrid intervening sequence. We show that in this
system a vector with the CMV promoter, the tripartite leader sequence and
the intron, referred to as pMCl. is the best of the examined combinations.
Using conventional tissue culturing of CHO cells stably transfected with
this vector, we were able to isolate glycosylated lysozyme with a yield of
4.5 mg per liter of spent medium. (C) 2002 Elsevier Science B.V. All
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L14 ANSWER 2 OF 3 MEDLINE on STN
ACCESSION NUMBER: 2001201488 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11190674
TITLE: Functions of **selectins**.
AUTHOR: Ley K
CORPORATE SOURCE: Department of Biomedical Engineering, University of
Virginia, Charlottesville, Virginia 22908, USA.
SOURCE: Results and problems in cell differentiation, (2001) 33
177-200. Ref: 192
Journal code: 0173555. ISSN: 0080-1844.
PUB. COUNTRY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)

LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200104
ENTRY DATE: Entered STN: 20010417
Last Updated on STN: 20010417
Entered Medline: 20010412

AB The **selectins** are cell surface lectins that have evolved to mediate the adhesion of white blood cells to endothelial cells and platelets under flow. They recognize fucosylated, sialylated and in some cases sulfated ligands **expressed** on scaffold glycoproteins serving as functional counter-receptors. **Selectins** are regulated at the transcriptional level, through proteolytic processing, through cellular sorting, and through regulated **expression** of **glycosyl-transferases** responsible for the formation of functional ligands. The **selectins** are physiologically important in inflammation, lymphocyte homing, immunological responses, and homing of bone marrow stem cells. They play a role in atherosclerosis, ischemia-reperfusion injury, inflammatory diseases, and metastatic spreading of some cancers.

L14 ANSWER 3 OF 3 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1998:672098 HCAPLUS

DOCUMENT NUMBER: 130:37273

TITLE: Single glycosyltransferase, core 2
 β 1 \rightarrow 6-N-acetylglucosaminyltransferase,
regulates cell surface sialyl-LeX **expression**
level in **human** pre-B lymphocytic leukemia
cell line KM3 treated with phorbol ester

AUTHOR(S): Nakamura, Mitsuru; Kudo, Takashi; Narimatsu, Hisashi;
Furukawa, Yusuke; Kikuchi, Jiro; Asakura, Shinji;
Yang, Wei; Iwase, Satsuki; Hatake, Kiyohiko; Miura,
Yasusada

CORPORATE SOURCE: Division of Hemopoiesis, Jichi Medical School,
Tochigi, 329-04, Japan

SOURCE: Journal of Biological Chemistry (1998), 273(41),
26779-26789

CODEN: JBCHA3; ISSN: 0021-9258

PUBLISHER: American Society for Biochemistry and Molecular
Biology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Sialyl-LeX (sLeX) antigen **expression** recognized by KM93 monoclonal antibody was down-regulated during differentiation induced by 12-O-tetradecanoylphorbol-13-acetate (TPA) in **human** pre-B lymphocytic leukemia cell line KM3. The sLeX determinants were almost exclusively **expressed** on O-linked oligosaccharide chains of an O-glycosylated 150-kDa glycoprotein (gp150). A low shear force cell adhesion assay showed that TPA treatment inhibited E-**selectin**-mediated cell adhesion. Transcript and/or enzyme activity levels of α 1 \rightarrow 3-fucosyltransferase, α 2 \rightarrow 3-sialyltransferase, β 1 \rightarrow 4-galactosyltransferase, and elongation β 1 \rightarrow 3-N-acetylglucosaminyltransferase did not correlate with sLeX **expression** levels. However, transcript and enzyme activity levels of core 2 GlcNAc-transferase (C2GnT) were down-regulated during TPA treatment. Following transfection and constitutive **expression** of full-length exogenous C2GnT transcript, C2GnT enzyme activities were maintained at high levels even after TPA treatment and down-regulation of cell surface sLeX antigen **expression** by TPA was completely abolished. Furthermore, in the transfected cells, the KM93 reactivity of gp150 was not reduced by TPA treatment, and the inhibition of cell adhesion by TPA was also blocked. Thus, sLeX **expression** is critically regulated by a single **glycosyl-transferase**, C2GnT, during differentiation of KM3 cells.

REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s "glyCAM-1" or "CD34" or "MadCAM-1" or "Sgp200" or "podocalyxin"
 L15 63891 "GLYCAM-1" OR "CD34" OR "MADCAM-1" OR "SGP200" OR "PODOCALYXIN"

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 L14 3 DUP REM L13 (0 DUPLICATES REMOVED)
 L15 63891 S "GLYCAM-1" OR "CD34" OR "MADCAM-1" OR "SGP200" OR "PODOCALYXI"

=> s l9 and l15

L16 9 L9 AND L15

=> dup rem l16

PROCESSING COMPLETED FOR L16

L17 4 DUP REM L16 (5 DUPLICATES REMOVED)

=> d 1-4 ibib ab

L17 ANSWER 1 OF 4 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2003:379629 HCAPLUS

DOCUMENT NUMBER: 139:66640

TITLE: Transcription starting from an alternative promoter leads to the **expression** of the **human** ABO histo-blood group antigen

AUTHOR(S): Hata, Yukiko; Kominato, Yoshihiko; Takizawa, Hisao; Tabata, Sachiyo; Michino, Junko; Nishino, Kazuma; Yasumura, Satoshi; Yamamoto, Fumiichiro

CORPORATE SOURCE: Faculty of Medicine, Department of Legal Medicine, Toyama Medical and Pharmaceutical University, Japan

SOURCE: Transfusion (Malden, MA, United States) (2003), 43(5), 656-662

CODEN: TRANAT; ISSN: 0041-1132

PUBLISHER: Blackwell Publishing, Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Using the 5'-rapid amplification of cDNA ends technique with the ex vivo culture of AC133-**CD34+** cells, a transcription start site was recently identified approx. 0.7 kb upstream from the transcription start sites previously determined. The transcripts from the alternative starting exon 1a were demonstrated in the cells of both erythroid and epithelial lineages. Because the nucleotide sequence of exon 1a does not contain an ATG codon, we examined whether transcription starting from exon 1a leads to production of a functional **glycosyl-transferase**. Stable transfection expts. into the **human** gastric cancer MKN28 cells were performed using the various A transferase **expression**

plasmids. Large amts. of A antigens were demonstrated on the cells transfected with the A transferase **expression** plasmid containing the entire cDNA from exon 1a or the 5'-truncated cDNA leading to the production of the N-truncated protein with deletion of the cytoplasmic tail and a portion of the transmembrane domain. However, negligible amts. of A antigens were observed on the cells transfected with the A transferase **expression** plasmids containing the 5'-truncated cDNA leading to the production of the N-truncated proteins without the cytoplasmic tail and the transmembrane domain. This study suggests that a functional A transferase could be produced by the transcription from exon 1a.

REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 2 OF 4 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
 ACCESSION NUMBER: 2002:501531 BIOSIS
 DOCUMENT NUMBER: PREV200200501531
 TITLE: Microvascular architecture and cellular differentiation of solitary and multiple hepatic adenomas.
 AUTHOR(S): Theuerkauf, I. [Reprint author]; Puetz, U.; Axmann, C.; Fischer, H. P. [Reprint author]
 CORPORATE SOURCE: Institut fuer Pathologie, Rhein. Friedrich-Wilhelms Universitaet, Bonn, Germany
 SOURCE: Pathology Research and Practice, (2002) Vol. 198, No. 3, pp. 221. print.
 Meeting Info.: 86th Meeting of the German Society of Pathology. Vienna, Austria. April 03-06, 2002.
 CODEN: PARPDS. ISSN: 0344-0338.
 DOCUMENT TYPE: Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 LANGUAGE: English
 ENTRY DATE: Entered STN: 25 Sep 2002
 Last Updated on STN: 25 Sep 2002

L17 ANSWER 3 OF 4 MEDLINE on STN DUPLICATE 1
 ACCESSION NUMBER: 97281434 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 9135742
 TITLE: **Human CD34+** cells do not **express** glutathione S-transferases alpha.
 AUTHOR: Czerwinski M; Kiem H P; Slattey J T
 CORPORATE SOURCE: Department of Pharmaceutics, University of Washington, Seattle 98195-3576, USA.
 CONTRACT NUMBER: CA 18029 (NCI)
 HL 53750 (NHLBI)
 SOURCE: Gene therapy, (1997 Mar) 4 (3) 268-70.
 Journal code: 9421525. ISSN: 0969-7128.
 PUB. COUNTRY: ENGLAND: United Kingdom
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199706
 ENTRY DATE: Entered STN: 19970709
 Last Updated on STN: 19980206
 Entered Medline: 19970626

AB The **expression** of glutathione S-transferases alpha (**GST alpha**) in **human** hematopoietic **CD34+** cells and bone marrow was studied using RT-PCR and immunoblotting. The **GSTA1** protein conjugates glutathione to the stem cell selective alkylator busulfan. This reaction is the major pathway of elimination of the compound from the **human** body. **Human** hematopoietic **CD34+** cells and bone marrow do not **express** **GSTA1** message, which was present at a high level in liver, an organ relatively resistant to busulfan toxicity in comparison to bone marrow. Similarly, baboon **CD34+** cells and dog bone marrow do not **express** **GSTA1**. **Human** **GSTA1** may be useful as a chemoprotective

selectable marker in human stem cell gene therapy.

L17 ANSWER 4 OF 4 LIFESCI COPYRIGHT 2004 CSA on STN
ACCESSION NUMBER: 97:103916 LIFESCI
TITLE: Human CD34 super(+) cells do not
express glutathione S-transferases alpha
AUTHOR: Czerwinski, M.; Kiem, H.-P.; Slattery, J.T.*
CORPORATE SOURCE: Department of Pharmaceutics, Box 357610, University of
Washington, Seattle, WA 98195-3576, USA
SOURCE: GENE THER., (1997) vol. 4, no. 3, pp. 267-270.
ISSN: 0969-7128.
DOCUMENT TYPE: Journal
FILE SEGMENT: G; W3
LANGUAGE: English
SUMMARY LANGUAGE: English

AB The **expression** of glutathione S-transferases alpha (GST
alpha) in human hematopoietic CD34 super(+) cells and bone marrow was studied using RT-PCR and immunoblotting. The GSTA1 protein conjugates glutathione to the stem cell selective alkylator busulfan. This reaction is the major pathway of elimination of the compound from the human body. Human hematopoietic CD34 super(+) cells and bone marrow do not **express** GSTA1 message, which was present at a high level in liver, an organ relatively resistant to busulfan toxicity in comparison to bone marrow. Similarly, baboon CD34 super(+) cells and dog bone marrow do not **express** GSTA1. Human GSTA1 may be useful as a chemoprotective selectable marker in human stem cell gene therapy.

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L17 4 DUP REM L16 (5 DUPLICATES REMOVED)

=> s l5 and l7

L18 33 L5 AND L7

=> dup rem l18

PROCESSING COMPLETED FOR L18

L19 12 DUP REM L18 (21 DUPLICATES REMOVED)

=> d 1-12 ibib ab

L19 ANSWER 1 OF 12 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2002:391865 HCAPLUS
 DOCUMENT NUMBER: 136:397039
 TITLE: Protein and cDNA sequences of novel **human glycosyl transferase** sequence homologs and diagnostic and therapeutic uses thereof
 INVENTOR(S): Meyers, Rachel; Williamson, Mark
 PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., USA
 SOURCE: PCT Int. Appl., 153 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002040657	A2	20020523	WO 2001-US47575	20011120
WO 2002040657	A3	20031211		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
 RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2002026062	A5	20020527	AU 2002-26062	20011120
US 2002115628	A1	20020822	US 2001-1851	20011120
EP 1385940	A2	20040204	EP 2001-995484	20011120

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

PRIORITY APPLN. INFO.: US 2000-249939P P 20001120
 WO 2001-US47575 W 20011120

AB The invention provides protein and cDNA sequences of novel human proteins, designated 47169 and 33935, which have sequence homol. with glycosyl transferases. The invention also provides antisense nucleic acid mols., **recombinant expression** vectors containing 47169 and 33935 nucleic acid mols., host cells into which the **expression** vectors have been introduced, and non-human transgenic animals in which a 47169 or 33935 gene has been introduced or disrupted. The invention still further provides isolated 47169 and 33935 proteins, fusion proteins, antigenic peptides and anti-47169 and anti-33935 antibodies. Diagnostic methods utilizing compns. of the invention are also provided.

L19 ANSWER 2 OF 12 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2003:779656 HCAPLUS
 DOCUMENT NUMBER: 139:272110
 TITLE: Protein and cDNA sequences of 25.52-kilodalton **human glycosyl transferase** sequence homolog and their therapeutic uses
 INVENTOR(S): Mao, Yumin; Xie, Yi
 PATENT ASSIGNEE(S): Bode Gene Development Co., Ltd., Peop. Rep. China
 SOURCE: Faming Zhuanli Shenqing Gongkai Shuomingshu, 31 pp.
 CODEN: CNXXEV
 DOCUMENT TYPE: Patent
 LANGUAGE: Chinese
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
CN 1380335	A	20021120	CN 2001-105934	20010410
PRIORITY APPLN. INFO.:			CN 2001-105934	20010410

AB The invention provides protein and cDNA sequences of a novel 25.52-kilodalton human protein, designated as "glycosyl transferase 25.52", which is homologous to glycosyl transferase. The invention relates to **expression** of glycosyl transferase sequence homolog in E. coli BL21(DE3)plySs transfected with plasmid pET-28(+). The invention also relates to preparation of antibody against glycosyl transferase sequence homolog. The invention further relates to the uses of the glycosyl transferase sequence homolog in treatment of glycosyl transferase-related diseases.

L19 ANSWER 3 OF 12 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2001:731018 HCAPLUS

DOCUMENT NUMBER: 135:268368

TITLE: Protein and cDNA sequences of novel **human glycosyl transferase** sequence homologs and uses thereof

INVENTOR(S): Meyers, Rachel A.

PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., USA

SOURCE: PCT Int. Appl., 136 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 14

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001073049	A2	20011004	WO 2001-US9358	20010322
WO 2001073049	A3	20020516		
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG			
US 2003224376	A1	20031204	US 2002-184648	20020627
PRIORITY APPLN. INFO.:			US 2000-191964P	P 20000324
			US 2000-187456P	P 20000307
			US 2000-191865P	P 20000324
			US 2000-192092P	P 20000324
			US 2000-199500P	P 20000425
			US 2000-200604P	P 20000428
			US 2000-205408P	P 20000519
			US 2000-211730P	P 20000615
			US 2000-212077P	P 20000615
			US 2000-212079P	P 20000615
			US 2000-235044P	P 20000925
			US 2000-238849P	P 20001006
			US 2001-267494P	P 20010208
			US 2001-801220	A2 20010307
			WO 2001-US7269	A 20010307
			US 2001-815028	A2 20010322
			WO 2001-US9358	A 20010322
			US 2001-816714	B2 20010323
			WO 2001-US9468	A 20010323
			US 2001-817910	A2 20010326
			WO 2001-US9633	A 20010326
			US 2001-842528	B2 20010425
			WO 2001-US40607	A 20010425
			US 2001-844948	A2 20010427
			WO 2001-US13805	A 20010427

US 2001-861164	B2 20010518
WO 2001-US16292	A 20010518
US 2001-882836	A2 20010615
US 2001-882872	B2 20010615
US 2001-883060	A2 20010615
WO 2001-US19138	A 20010615
WO 2001-US19153	A 20010615
WO 2001-US19543	A 20010615
US 2001-962678	A2 20010925
WO 2001-US29963	A 20010925
US 2001-973457	A2 20011009
US 2002-72285	A2 20020208
WO 2002-US3736	A 20020208

AB The invention provides protein and cDNA sequences of novel human proteins, designated 33877 and 47179, which have sequence homol. with glycosyltransferase members. The invention also provides antisense nucleic acid mols., **recombinant expression** vectors containing 33877 or 47179 nucleic acid mols., host cells into which the **expression** vectors have been introduced, and nonhuman transgenic animals in which a 33877 or 47179 gene has been introduced or disrupted. The invention still further provides isolated 33877 or 47179 proteins, fusion proteins, antigenic peptides and anti-33877 or 47179 antibodies. Diagnostic methods utilizing compns. of the invention are also provided.

L19 ANSWER 4 OF 12 MEDLINE on STN DUPLICATE 1
 ACCESSION NUMBER: 2001038213 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 10934196
 TITLE: Modulation of glutathione S-transferase alpha by hepatitis B virus and the chemopreventive drug oltipraz.
 AUTHOR: Jaitovitch-Groisman I; Fotouhi-Ardakani N; Schechter R L; Woo A; Alaoui-Jamali M A; Batist G
 CORPORATE SOURCE: Lady Davis Institute of the Sir Mortimer B. Davis Jewish General Hospital, The Center for Translational Research in Cancer, Department of Medicine, McGill University, Montreal, Quebec H3T 1E2, Canada.
 SOURCE: Journal of biological chemistry, (2000 Oct 27) 275 (43) 33395-403.
 Journal code: 2985121R. ISSN: 0021-9258.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200011
 ENTRY DATE: Entered STN: 20010322
 Last Updated on STN: 20010322
 Entered Medline: 20001124

AB Persistent infection by hepatitis B virus (HBV) and exposure to chemical carcinogens correlates with the prevalence of hepatocellular carcinoma in endemic areas. The precise nature of the interaction between these factors is not known. Glutathione S-transferases (GST) are responsible for the cellular metabolism and detoxification of a variety of cytotoxic and carcinogenic compounds by catalysis of their conjugation with glutathione. Diminished GST activity could enhance cellular sensitivity to chemical carcinogens. We have investigated GST isozyme **expression** in hepatocellular HepG2 cells and in an HBV-transfected subline. Total GST activity and selenium-independent glutathione peroxidase activity are significantly decreased in HBV transfected cells. On immunoblotting, HBV transfected cells demonstrate a significant decrease in the level of GST Alpha class. Cytotoxicity assays reveal that the HBV transfected cells are more sensitive to a wide range of compounds known to be detoxified by GST Alpha conjugation. Although no significant difference in protein half-life between the two cell lines was found, semi-quantitative reverse transcription-polymerase chain reaction shows a reduced amount of GST Alpha mRNA in the transfected cells. Because the

HBV x protein (HBx) seems to play a role in HBV transfection, we also demonstrated that **expression** of the HBx gene into HepG2 cells decreased the amount of GST Alpha protein. Transient transfection experiments using both rat and **human GST Alpha** (rGSTA5 and hGSTA1) promoters in HepG2 cells show a decreased CAT activity upon HBx **expression**, supporting a transcriptional regulation of both genes by HBx. This effect is independent of HBx interaction with Sp1. Treatment with oltipraz, an inducer of GST Alpha, partially overcomes the effect of HBx on both promoters. Promoter deletion studies indicate that oltipraz works through responsive elements distinct from AP1 or NF-kappaB transcription factors. Thus, HBV infection alters phase II metabolizing enzymes via different mechanisms than those modulated by treatment with oltipraz.

L19 ANSWER 5 OF 12 LIFESCI COPYRIGHT 2004 CSA on STN
 ACCESSION NUMBER: 2000:111488 LIFESCI
 TITLE: Differential Binding Affinities of PCBs, HO-PCBs, and Aroclors with **Recombinant** Human, Rainbow Trout (Oncorhynchus mykiss), and Green Anole (Anolis carolinensis) Estrogen Receptors, Using a Semi-High Throughput Competitive Binding Assay
 AUTHOR: Mathews, J.; Zacharewski, T.
 CORPORATE SOURCE: Department of Biochemistry and National Food Safety and Toxicology Center, Michigan State University, East Lansing, Michigan 48824-1319, USA
 SOURCE: Toxicological Sciences [Toxicol. Sci.], (20000200) vol. 53, no. 2, pp. 326-339.
 ISSN: 1096-6080.
 DOCUMENT TYPE: Journal
 FILE SEGMENT: X
 LANGUAGE: English
 SUMMARY LANGUAGE: English

AB A comparative study was undertaken to assess the ability of 44 polychlorinated biphenyls (PCBs), 9 hydroxylated PCBs (HO-PCBs), and 8 aroclors at concentrations ranging from 1 nM to 10 μ M to compete with [³H]17 beta -estradiol (E2) for binding to bacterially **expressed** fusion proteins using a semi-high throughput competitive-binding assay. The fusion proteins consisted of the D, E, and F domains of human (alpha), **cloned** reptilian (Anolis carolinensis) and recloned rainbow trout (Oncorhynchus mykiss) estrogen receptors (ER) linked to the glutathione S-transferase (GST) protein. GST-hER alpha def (**human**), **GST- alpha** ERdef (reptile) and GST-rtERdef (rainbow trout) fusion proteins exhibited high affinity for E2 with dissociation constants ($K_{sub(d)}$) of 0.4 plus or minus 0.1 nM, 0.7 plus or minus 0.2 nM, and 0.6 plus or minus 0.1 nM, respectively. Of the 44 PCBs examined, only PCBs 104, 184, and 188 effectively competed with [³H]E2 for binding to the GST-rtERdef protein with IC_{sub(50)} values ranging from 0.4-1.3 μ M. In contrast, these same congeners only caused a 30% displacement of [³H]E2 in GST-hER alpha def and GST- alpha ERdef proteins. Several additional congeners were found to bind to the GST-rtERdef fusion protein, although the degree of interaction varied among congeners. Among the HO-PCBs, 2',3',4',5'-tetrachloro-4-biphenylol and 2,6,2',6'-tetrachloro-4-biphenylol bound to all three fusion proteins with IC_{sub(50)} values ranging from 0.1-0.3 μ M. Dimethyl sulphoxide (DMSO) concentrations of 20% significantly increased the ability of PCBs 104, 184, and 188 to compete with [³H]E2 for binding to the GST-ERdef fusion proteins, whereas at 20% DMSO, a significant reduction in saturable binding was observed. These results demonstrate that ERs from different species exhibit differential ligand preferences and relative binding affinities for PCBs, which can be dramatically affected by DMSO concentration.

L19 ANSWER 6 OF 12 MEDLINE on STN DUPLICATE 2
 ACCESSION NUMBER: 2000174772 MEDLINE

DOCUMENT NUMBER: PubMed ID: 10711630
TITLE: The influence of diet on the regional distribution of glutathione S-transferase activity in channel catfish intestine.
AUTHOR: Gadagbui B K; James M O
CORPORATE SOURCE: Department of Medicinal Chemistry, College of Pharmacy, University of Florida, Gainesville 32610-0485, USA.
CONTRACT NUMBER: ES-05781 (NIEHS)
ES-07375 (NIEHS)
SOURCE: Journal of biochemical and molecular toxicology, (2000) 14 (3) 148-54.
Journal code: 9717231. ISSN: 1095-6670.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200003
ENTRY DATE: Entered STN: 20000407
Last Updated on STN: 20000407
Entered Medline: 20000324

AB There is evidence that glutathione conjugates are the major metabolites formed following systemic uptake of carcinogenic contaminants from the intestine. The effect of commercial diet versus a semi-purified diet on the distribution of glutathione S-transferase (GST) activity was examined in proximal, medial, and distal sections of catfish intestine. The bulk of GST activity with 1-chloro-2,4-dinitrobenzene, ethacrynic acid, and 3H-benzo[a]pyrene-4,5-oxide, and the percent cytosolic protein cross-reacting with anti-catfish GST-pi were in the more proximal segments and dropped off distally in the two diet groups. However, the total GST-pi cross-reacting protein in the proximal section was significantly higher in fish fed a chow diet. Western blot analysis revealed pi-class GST to be **expressed** principally in the proximal intestine. Cytosol samples cross-reacted with antibodies to **human GST-alpha**, -mu, and -pi, but not -theta, classes. Alpha-like GST isoforms of MW 26,200 and 24,600, absent in sections from fish fed a purified diet, were differentially **expressed** only in the distal section of chow-fed fish. These results indicate that diet significantly elicits regional differences in GST protein levels, that components of the commercial chow affect GST protein **expression** in the distal intestine, and that maintenance diet should be taken into consideration during dietary exposure studies.

L19 ANSWER 7 OF 12 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 3

ACCESSION NUMBER: 1998339046 EMBASE
TITLE: A 47-amino-acid fragment of SV40 T antigen represses transcription from **human GST-alpha** promoters.
AUTHOR: Sompayrac L.; Jane S.; Lorper M.; Sies H.
CORPORATE SOURCE: L. Sompayrac, Molec. Cellular,/Devtl. Biol. Dept., University of Colorado, Boulder, CO 80309, United States. laurens@Alum.mit.edu
SOURCE: Virology, (30 Sep 1998) 249/2 (275-285).
Refs: 32
ISSN: 0042-6822 CODEN: VIRLAX
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
LANGUAGE: English
SUMMARY LANGUAGE: English
AB SV40 T antigen downregulates the **expression** of an important detoxication enzyme, glutathione S-transferase α (GST α). We show here that the target of this repression is a 14-bp element common to the human GSTA1 and GSTA2 promoters. This element, which we have named

TAGR, is also critical for high-level, constitutive **expression** from these promoters. The TAGR element does not appear to contain a binding site for any transcription factor known to be present in fibroblasts, although the TAGR element does resemble the binding site for the Ikaros transcription factor found in hematopoietic cells. We also have identified a 47-amino-acid fragment of T antigen that includes amino acids 83-100 and 119-147, which is sufficient to repress transcription from the GST α promoter in transient transcription assays. Thus, GST α repression does not require binding of T antigen to pRb, p300, or p53, since the domains of T antigen required for binding these cellular proteins are missing from this T antigen fragment. We show, however, that this fragment does bind to three cellular proteins with approximate molecular weights of 54, 59, and 94 kDa.

L19 ANSWER 8 OF 12 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
 ACCESSION NUMBER: 1998:834534 SCISEARCH
 THE GENUINE ARTICLE: 132NF
 TITLE: Identification of two activating elements in the proximal promoter region of the human glutathione transferase-A1 and -A2 genes
 AUTHOR: Lorper M; Clairmont A; Carlberg C; Sies H (Reprint)
 CORPORATE SOURCE: UNIV DUSSELDORF, INST PHYSIOL CHEM 1, POSTFACH 10 10 07, D-40001 DUSSELDORF, GERMANY (Reprint); UNIV DUSSELDORF, INST PHYSIOL CHEM 1, D-40001 DUSSELDORF, GERMANY; UNIV DUSSELDORF, BIOL MED FORSCHUNGSZENTRUM, D-40001 DUSSELDORF, GERMANY
 COUNTRY OF AUTHOR: GERMANY
 SOURCE: ARCHIVES OF BIOCHEMISTRY AND BIOPHYSICS, (1 NOV 1998) Vol. 359, No. 1, pp. 122-127.
 Publisher: ACADEMIC PRESS INC JNL-COMP SUBSCRIPTIONS, 525 B ST, STE 1900, SAN DIEGO, CA 92101-4495.
 ISSN: 0003-9861.
 DOCUMENT TYPE: Article; Journal
 FILE SEGMENT: LIFE
 LANGUAGE: English
 REFERENCE COUNT: 21

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Promoter regions derived from the human glutathione S-transferase (GST) alpha gene cluster located on chromosome 6p12 were studied: the identical proximal promoters of the GST A1 and GST A2 genes and a proximal promoter of a pseudogene of this class. The sequence of the pseudogene promoter differs in four single nucleotides at positions -86, -66, -41, and -13, and a noncritical TTT insertion at positions -71 to -69 from the GST A1/A2 promoter. Here, it was shown that the GST A1/A2 proximal promoters differed by a factor of 3.4 in their activity from the proximal pseudogene promoter. Therefore, the functional significance of single base exchanges was examined by introducing individual point mutations at the four positions within the proximal GST A1/A2 promoter. In functional tests in transiently transfected human hepatoblastoma HepG2 cells the base exchange at position -13 showed no effect, whereas mutations at position -41 or -86 diminished the promoter activity to a level comparable to the pseudogene promoter. Promoter fragments of both genes spanning over these four sites were analyzed in a heterologous promoter context for their functionality in HepG2 cells. Moreover, gel shift experiments showed specific binding of nuclear proteins to these promoter fragments. The results show that in the proximal GST A1/A2 promoter the sites at position -41 or -86 are essential for the binding of activating transcription factor complexes. (C) 1998 Academic Press.

L19 ANSWER 9 OF 12 HCAPLUS COPYRIGHT 2004 ACS on STN
 ACCESSION NUMBER: 1998:434914 HCAPLUS
 DOCUMENT NUMBER: 129:199061
 TITLE: An attempt to predict the response of human glutathione S-transferase (GST) to chemical inducers

AUTHOR(S): using transgenic rats harboring human GST gene
Manabe, Sunao; Ando, Yosuke; Ohashi, Yoshihiko;
Igarashi, Isao; Yamoto, Takashi; Takaoka, Masaya;
Tanase, Hisao; Matsunuma, Naohika; Suzuki, Takashige;
Itoh, Kazumi
CORPORATE SOURCE: Laboratory Animal Science and Toxicology Laboratories,
Sankyo Co., Ltd, Fukuroi, 437, Japan
SOURCE: Journal of Toxicologic Pathology (1997), 10(3),
133-136
CODEN: JTPAE7; ISSN: 0914-9198
PUBLISHER: Japanese Society of Toxicologic Pathology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB To study the response of human glutathione S-transferase (GST) to chemical inducers, we have developed a line of transgenic rats which harbor 4.5 kb of **human GST alpha 1** promoter region in their genome. This promoter is linked to the chloramphenicol acetyltransferase (CAT) reporter gene which allows determination of the **expression** of human GST in rat tissues. Three chemical inducers, which show clearly different induction profiles, phenobarbital (PB), β -naphthoflavone (BNF), and butylated hydroxyanisole (BHA), were administered to the transgenic rats. Induction of constitutive rat liver enzymes by the inducers, which was evaluated in terms of the activities of P 450, GST, and UDP-glucuronosyltransferase in the liver tissues, were in agreement with what has been reported for non-transgenic rats. **Expression** of CAT protein was detected in the liver of the transgenic rats, and an unequivocal increase in CAT protein was found in the transgenic rats treated with PB. No remarkable changes in CAT protein were observed in the transgenic rats treated with BNF or BHA. Moreover, immunohistochem. staining with anti-CAT antibody revealed that the **expression** and increase of CAT protein were localized in the central zone of the liver lobule. The results of this study suggest that **human GST alpha 1** is induced by PB, in particular, in the central zone of the liver lobule. The transgenic rat is concluded to be a useful animal model for predicting metabolizing functions in humans.

REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L19 ANSWER 10 OF 12 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN

ACCESSION NUMBER: 96:197184 SCISEARCH

THE GENUINE ARTICLE: TY855

TITLE: SANDWICH ELISA FOR GLUTATHIONE-S-TRANSFERASE ALPHA1-1 -
PLASMA-CONCENTRATIONS IN CONTROLS AND IN PATIENTS WITH
GASTROINTESTINAL DISORDERS

AUTHOR: MULDER T P J (Reprint); PETERS W H M; COURT D A; JANSEN J
B M J

CORPORATE SOURCE: UNIV NIJMEGEN ST RADBOUD HOSP, DEPT GASTROENTEROL &
HEPATOL, POB 9101, 6500 HB NIJMEGEN, NETHERLANDS (Reprint)

COUNTRY OF AUTHOR: NETHERLANDS

SOURCE: CLINICAL CHEMISTRY, (MAR 1996) Vol. 42, No. 3, pp. 416-419

ISSN: 0009-9147.

DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE

LANGUAGE: ENGLISH

REFERENCE COUNT: 20

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Class Alpha glutathione S-transferases (GST-Alpha) are found in high concentrations in human liver. Increased plasma concentrations of GSTA1-1, the most abundant isoform of GST-Alpha, are a very sensitive marker for hepatocellular leakage. A sandwich-type ELISA was developed, based on a monoclonal antibody specific for human GSTA1-1 and a polyclonal rabbit anti-human GST-Alpha antiserum. The assay is

specific for human GSTA1-1, and has a detection limit of 0.04 μ g/L. The distribution of plasma GSTA1-1 concentrations in 350 blood donors was nearly normalized by logarithmic transformation and an upper normal reference concentration of 5.9 μ g/L was calculated. Men had significantly higher plasma GSTA1-1 concentrations than women ($P < 0.0001$). In women, but not in men, a significant increase was noted with age ($P < 0.05$). In patients with inflammatory bowel disease ($n = 210$), gastrointestinal tumors ($n = 70$), irritable bowel disease ($n = 36$), or chronic pancreatitis ($n = 12$), plasma GSTA1-1 concentrations were similar to those of controls. In contrast, plasma GSTA1-1 concentrations were increased to a similar extent as alanine aminotransferase activities in patients with liver disorders ($n = 37$).

L19 ANSWER 11 OF 12 MEDLINE on STN DUPLICATE 4
 ACCESSION NUMBER: 95262669 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 7744032
 TITLE: Turnover of glutathione S-transferase alpha mRNAs is accelerated by 12-O-tetradecanoyl phorbol-13-acetate in human hepatoma and colon carcinoma cell lines.
 AUTHOR: Eickelmann P; Morel F; Schulz W A; Sies H
 CORPORATE SOURCE: Institut fur Physiologische Chemie I, Heinrich-Heine-Universitat, Dusseldorf, Germany.
 SOURCE: European journal of biochemistry / FEBS, (1995 Apr 1) 229 (1) 21-6.
 Journal code: 0107600. ISSN: 0014-2956.
 PUB. COUNTRY: GERMANY: Germany, Federal Republic of
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199506
 ENTRY DATE: Entered STN: 19950621
 Last Updated on STN: 19980206
 Entered Medline: 19950615

AB The phorbol ester, 12-O-tetradecanoyl phorbol-13-acetate (TPA), known to induce murine glutathione S-transferase (GST) Ya, was examined for its effect on the **expression of human GST alpha**. Unexpectedly, 24-h treatment of the human hepatoma cell line HepG2 with 100 nmol/l TPA caused a decrease of the GST alpha mRNA level to below 5% of controls, i.e. opposite to the known response in the mouse. The level of mRNA for GST Mu was also decreased, but the mRNAs of c-jun and jun-B were elevated after 2 h. The decrease of GST alpha mRNAs was inhibited by staurosporine, suggesting an involvement of protein kinase C. Inhibition of transcription and translation by actinomycin D and cycloheximide also partially inhibited the effect of TPA on the **expression of GST alpha**. In the presence of actinomycin D, GST alpha mRNA half-life was 14.5 h, compared to 3.5 h in the presence of TPA. The calcium ionophore A23187 caused a loss of GST alpha mRNAs to levels almost as low as those obtained with TPA. The effects of TPA and the calcium ionophore were also observed in CaCo2 colon carcinoma cells. As a consequence of the decrease of mRNA levels, GST alpha protein levels and total GST enzyme activity were also diminished. Also, the morphology of the cells was changed after 3 h exposure to TPA. These data suggest that **human GST alpha expression** can be regulated at the level of mRNA stability by a pathway involving protein kinase C.

L19 ANSWER 12 OF 12 MEDLINE on STN DUPLICATE 5
 ACCESSION NUMBER: 94291255 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 8020149
 TITLE: Protection by transfected glutathione S-transferase isozymes against carcinogen-induced alkylation of cellular macromolecules in human MCF-7 cells.
 AUTHOR: Fields W R; Li Y; Townsend A J
 CORPORATE SOURCE: Biochemistry Department, Bowman Gray School of Medicine,

Wake Forest University Comprehensive Cancer Center,
Winston-Salem, NC 27157.
CONTRACT NUMBER: 5F31GM14822-02 (NIGMS)
R-55-ES-06006-01 (NIEHS)
SOURCE: Carcinogenesis, (1994 Jun) 15 (6) 1155-60.
Journal code: 8008055. ISSN: 0143-3334.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199408
ENTRY DATE: Entered STN: 19940815
Last Updated on STN: 19980206
Entered Medline: 19940803

AB Increased **expression** of glutathione S-transferase (GST) isozymes has been correlated with development of resistance both to cytotoxic anticancer agents and to genotoxic carcinogens. While most anticancer agents are poor GST substrates, the model alkylating carcinogen 4-nitroquinoline-1-oxide (NQO) is a good substrate for human pi class GST (hGSTP1-1) and murine GST mu-1 (mGSTM1-1), but not **human GST alpha-2** (hGSTA2-2). We investigated whether **expression** of these GST isozymes in stably transfected **clonal** cell lines could protect against the genotoxic and cytotoxic effects of NQO. Compared to parental MCF-7 or pSV2neotransfected control cell lines, covalent labeling of total cellular macromolecules by [3H]NQO (0.1-1.0 mM) was reduced by 70% and 92% in hGSTP1-1- and mGSTM1-1-transfected cell lines, respectively, but was not affected in the hGSTA2-2 **expressing** line. The observed protection was closely correlated with the relative specific activity of each cell line for conjugation of NQO by the transfected GST isozymes and this protection was reversible by pretreatment of cells with the GST inhibitor ethacrynic acid. Similar results were obtained when covalent labeling of total cellular nucleic acid or DNA was measured. However, **clonogenic** survival assays indicated that the sensitivity of these cell lines to the cytotoxic effects of NQO was similar for the control and GST-transfected MCF-7 cell lines. Thus, while **expression** of hGSTP1-1 and mGSTM1-1 (but not hGSTA2-2) was highly protective against alkylation of cellular macromolecules by NQO, this protection was not effective against cytotoxicity induced by NQO as measured by **clonogenic** assay. These results indicate that **expression** of GST isozymes can protect differentially against the acute genotoxic and potentially mutagenic effects, as compared to the cytotoxic effects, of electrophiles that are detoxified by glutathione conjugation.

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E1	1	ROSEN RT/AU
E2	2	ROSEN RUDOLPH A/AU
E3	2426 -->	ROSEN S/AU
E4	20	ROSEN S A/AU
E5	8	ROSEN S B/AU
E6	150	ROSEN S C/AU
E7	759	ROSEN S D/AU
E8	8	ROSEN S D */AU
E9	1	ROSEN S D C/AU
E10	53	ROSEN S E/AU
E11	13	ROSEN S F/AU
E12	145	ROSEN S G/AU

=> s e3

L20 2426 "ROSEN S"/AU

=> d his

(FILE 'HOME' ENTERED AT 11:06:03 ON 20 JUL 2004)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS,
LIFESCI' ENTERED AT 11:06:28 ON 20 JUL 2004

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L1      2714 S GLYCOSYL(A)TRANSFERASE?
L2      1105 S "GST-ALPHA" OR GST4(W)ALPHA
L3      3819 S L1 OR L2
L4      1242 S HUMAN AND L3
L5       50 S HUMAN(W)L3
L6       83 S HUMAN(2W)L3
L7     6615667 S CLON? OR EXPRESS? OR RECOMBINANT
L8      1242 S L4 OR L5 OR L6
L9       700 S L7 AND L8
L10     14569 S L(W)SELECTIN OR "L-SELECTIN"
L11       0 S L9 AND L10
L12     55768 S SELECTIN
L13       3 S L9 AND L12
L14       3 DUP REM L13 (0 DUPLICATES REMOVED)
L15     63891 S "GLYCAM-1" OR "CD34" OR "MADCAM-1" OR "SGP200" OR "PODOCALYXI
L16       9 S L9 AND L15
L17       4 DUP REM L16 (5 DUPLICATES REMOVED)
L18      33 S L5 AND L7
L19      12 DUP REM L18 (21 DUPLICATES REMOVED)
          E ROSEN S/AU
L20     2426 S E3
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=> s l3 and l20

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L21       0 L3 AND L20
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=> d 1-6 ibib ab

L25 ANSWER 1 OF 6 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2002:251882 HCAPLUS

DOCUMENT NUMBER: 136:291000

TITLE: Screening of novel **human glycosyl sulfotransferase** expressed in high endothelial cells (HEC) (GST-3, HEC-GlcNAc6ST) inhibitors

INVENTOR(S): Bistrup, Annette; Rosen, Steven D.; Tangemann, Kirsten; Hemmerich, Stefan

PATENT ASSIGNEE(S): The Regents of the University of California, USA

SOURCE: U.S., 38 pp., Cont.-in-part of U.S. Ser. No. 45,284.

CODEN: USXXAM

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6365365	B1	20020402	US 1998-190911	19981112
US 6265192	B1	20010724	US 1998-45284	19980320
CA 2322779	AA	19990930	CA 1999-2322779	19990226
WO 9949018	A1	19990930	WO 1999-US4316	19990226
W:	AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW:	GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG			
AU 9927945	A1	19991018	AU 1999-27945	19990226
AU 764852	B2	20030904		
EP 1062326	A1	20001227	EP 1999-908538	19990226
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI			
JP 2002507409	T2	20020312	JP 2000-537979	19990226
US 2001051370	A1	20011213	US 2001-816825	20010322
US 2002164748	A1	20021107	US 2001-7262	20011108
PRIORITY APPLN. INFO.:			US 1998-45284	A2 19980320
			US 1998-190911	A 19981112
			WO 1999-US4316	W 19990226

AB Use of a novel **human glycosyl sulfotransferase** expressed in high endothelial cells (HEC) (GST-3 or HEC-GlcNAc6ST) for screening inhibitors as therapeutic agent is provided. Full-length cDNAs containing the two contigs and predicting CS6T/KSST homologs were obtained by screening a human fetal brain λ ZAP cDNA library (Stratagene, La Jolla, Calif.) with labeled 700-800 bp restriction fragments (from EST 2 for contig 1 and from EST 5 for contig 2). The proteins encoded by these cDNAs were designated as GST 1 and GST 2, where GST denotes "glycosylsulfotransferase." GST 1 has been independently cloned and assigned the name "KSGal6ST by Fukuta et al., J. Biol. Chemical (1997) 272: 32321-8. ESTs potentially coding for novel **human glycosyl sulfotransferases** other than GST-1&2 were identified through a secondary homol. screen, in which the peptide sequences of GST-1 and GST-2 were used as template in two parallel TBLASTN searches against a public (dbest) and a private genomic database (Lifeseq, Incyte Pharmaceuticals, Palo Alto, Calif.). Three cDNA clones which encode three different human homologs for C6ST/KSST have been obtained. The predicted GST proteins are type 2 membrane proteins 411, 484, and 386 amino acids in length, resp. Each has a relatively short transmembrane

domain and a short amino terminal cytoplasmic tail. GST-1 is the same as the sulfotransferase reported by Fukuta et al. supra (1997) and named KSGal6ST. GST-3 (HEC-GlcNAc6ST), is a novel GlcNAc-6-sulfotransferase. The novel human glycosylsulfotransferase enzyme of the subject invention has been named **human glycosyl sulfotransferase** 3 or huGST-3 or HEC-GlcNAc6ST. HuGST-3 is capable of sulfating selectin ligands, particularly L-selectin ligands, e.g., GlyCAM-1. Donor compds. from which huGST-3 obtains sulfate groups for transfer to acceptor ligand compds. include 3'-phosphoadenosine 5'-phosphosulfate (PAPS) and the like. Selectin ligands capable of being sulfated through huGST-3 action include E-, P- and L-selectin ligands, particularly L-selectin ligands, such as GlyCAM-1, CD34, MadCAM-1, Sgp200, podocalyxin, and the like. huGST-3 is strongly predicted to have GlcNAc6-O-sulfotransferase (N-acetylglucosamine-6-O-sulfotransferase) activity. Human GST-3 is a 386 amino acid protein having an amino acid sequence as shown in FIG. 1 and identified as SEQ ID NO:01.

REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L25 ANSWER 2 OF 6 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2001:64196 HCAPLUS

DOCUMENT NUMBER: 134:127828

TITLE: Cloning of nucleic acid sequences encoding
**human and murine glycosyl
sulfotransferases**

INVENTOR(S): Rosen, Steven D.; Lee, Jin Kyu; Hemmerich, Stefan

PATENT ASSIGNEE(S): Regents of the University of California, USA

SOURCE: PCT Int. Appl., 128 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001006015	A1	20010125	WO 2000-US19741	20000719
W: AU, CA, JP				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 1210455	A1	20020605	EP 2000-948806	20000719
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				
JP 2003505039	T2	20030212	JP 2001-511223	20000719
PRIORITY APPLN. INFO.:				
			US 1999-144694P	P 19990720
			US 2000-593828	A 20000713
			WO 2000-US19741	W 20000719

AB Novel glycosyl sulfotransferases (GST-4 α , GST-4 β , and GST-6 from human; GST-4 and GST-6 from mouse) and polypeptides related thereto, as well as nucleic acid compns. encoding the same, are provided. The glycosyl sulfotransferases are type 2 membrane proteins having a relatively short transmembrane domain and N-terminal cytoplasmic tail of varying length, and are capable of sulfating selectin ligands, particularly L-selectin ligands (e.g., GlyCAM-1). Genomic DNA sequences encoding human GST-4 and GST-6 and for mouse GST-6 are also provided. The subject polypeptides and nucleic acid compns. find use in a variety of applications, including various diagnostic and therapeutic agent screening applications. Also provided are methods of inhibiting selectin-mediated binding events and methods of treating disease conditions associated therewith, particularly by administering an inhibitor of at least one of GST-4 α , GST-4 β , and GST-6.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L25 ANSWER 3 OF 6 BIOTECHDS COPYRIGHT 2004 THOMSON DERWENT/ISI on STN

ACCESSION NUMBER: 2000-00104 BIOTECHDS

TITLE: **Human** and mouse **glycosyl-sulfotransferase-3** and related polynucleotides; expression in mammalian host cell and antibody, used for disease diagnosis and gene therapy

AUTHOR: Bistrup A; Rosen S D; Tangemann K; Hemmerich S

PATENT ASSIGNEE: Univ.California; Syntex

LOCATION: Oakland, CA, USA; Palo Alto, CA, USA.

PATENT INFO: WO 9949018 30 Sep 1999

APPLICATION INFO: WO 1999-US4316 26 Feb 1999

PRIORITY INFO: US 1998-190911 12 Nov 1998; US 1998-45284 20 Mar 1998

DOCUMENT TYPE: Patent

LANGUAGE: English

OTHER SOURCE: WPI: 1999-580442 [49]

AB Glycosyl-sulfotransferase-3 (GST-3, 386 or 388 amino acids) present in other than its natural environment, is new. Also claimed are: a nucleic acid (2,032 or 1,893 bp) which encodes GST-3; an expression cassette under the control of initiation sequences and termination sequences; a host cell; a method of producing GST-3; a monoclonal antibody; a method for inhibiting the binding of a selectin and a selectin ligand; a method of inhibiting a selectin mediated binding event in a mammalian host; a method of modulating a symptom of a disease condition associated with a selectin mediated binding event; a method of diagnosing a disease state related to the abnormal levels of a sulfotransferase chosen from GST-3 and KSGal6ST; a method of determining whether an agent is capable of modulating the activity of a sulfotransferase chosen from GST-3 and KSGal6ST; and a non-human transgenic animal model for *gst-3* gene function. The nucleic acid sequences, DNA probes and DNA primers derived from these, proteins and antibodies are useful in detecting homologs. The products are useful in the diagnosis of diseases associated with selectin binding interactions. (59pp)

L25 ANSWER 4 OF 6 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN

ACCESSION NUMBER: 1998:906629 SCISEARCH

THE GENUINE ARTICLE: 137GQ

TITLE: Cloning and characterization of a **human glycosyl sulfotransferase** that is restricted to high endothelial venules and confers expression of the L-selectin recognition epitope 6-sulfo sialyl Lewis X.

AUTHOR: Bistrup A (Reprint); Bakhta S; Tangemann K; Lee J K; Gunn M D; Belov Y Y; Kannagi R; Hemmerich S; Rosen S D

CORPORATE SOURCE: UNIV CALIF SAN FRANCISCO, SAN FRANCISCO, CA 94143; ROCHE BIOSCI, PALO ALTO, CA; AIICHI CAN RES INST, NAGOYA, AICHI, JAPAN

COUNTRY OF AUTHOR: USA; JAPAN

SOURCE: MOLECULAR BIOLOGY OF THE CELL, (NOV 1998) Vol. 9, Supp. [S], pp. 718-718.

Publisher: AMER SOC CELL BIOLOGY, PUBL OFFICE, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814.

ISSN: 1059-1524.

DOCUMENT TYPE: Conference; Journal

FILE SEGMENT: LIFE

LANGUAGE: English

REFERENCE COUNT: 0

L25 ANSWER 5 OF 6 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

ACCESSION NUMBER: 1999:17006 BIOSIS

DOCUMENT NUMBER: PREV199900017006

TITLE: Cloning and characterization of a **human glycosyl sulfotransferase** that is restricted to high endothelial venules and confers expression of the L-selectin recognition epitope 6-sulfo

AUTHOR(S): sialyl Lewis X.
 Bistrup, Annette [Reprint author]; Bakhta, Sunil;
 Tangemann, Kirsten; Lee, Jin Kyu; Gunn, Michael D.; Belov,
 Yevgeniy Y.; Kannagi, Reiji; Hemmerich, Stefan; Rosen,
 Steven D.
 CORPORATE SOURCE: Univ. Calif., San Francisco, CA, USA
 SOURCE: Molecular Biology of the Cell, (Nov., 1998) Vol. 9, No.
 SUPPL., pp. 124A. print.
 Meeting Info.: 38th Annual Meeting of the American Society
 for Cell Biology. San Francisco, California, USA. December
 12-16, 1998. American Society for Cell Biology.
 CODEN: MBCEEV. ISSN: 1059-1524.
 DOCUMENT TYPE: Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 LANGUAGE: English
 ENTRY DATE: Entered STN: 20 Jan 1999
 Last Updated on STN: 20 Jan 1999

L25 ANSWER 6 OF 6 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
 ACCESSION NUMBER: 1998:810754 SCISEARCH
 THE GENUINE ARTICLE: 130CC
 TITLE: Cloning and functional characterization of a **human**
glycosyl sulfotransferase, that is
 highly restricted to high endothelial venules and confers
 expression of the L-selectin recognition epitope 6-sulfo
 sialyl Lewis x.
 AUTHOR: Hemmerich S (Reprint); Bistrup A; Bakhta S; Gunn M D;
 Kannagi R; Rosen S D
 CORPORATE SOURCE: ROCHE BIOSCI, PALO ALTO, CA; UNIV CALIF SAN FRANCISCO, SAN
 FRANCISCO, CA 94143; AIICHI CANC RES INST, NAGOYA, AICHI,
 JAPAN
 COUNTRY OF AUTHOR: USA; JAPAN
 SOURCE: GLYCOBIOLOGY, (NOV 1998) Vol. 8, No. 11, pp. 29-29.
 Publisher: OXFORD UNIV PRESS, GREAT CLARENDON ST, OXFORD
 OX2 6DP, ENGLAND.
 ISSN: 0959-6658.
 DOCUMENT TYPE: Conference; Journal
 FILE SEGMENT: LIFE
 LANGUAGE: English
 REFERENCE COUNT: 0

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(FILE 'HOME' ENTERED AT 11:06:03 ON 20 JUL 2004)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS,
 LIFESCI' ENTERED AT 11:06:28 ON 20 JUL 2004

L1 2714 S GLYCOSYL(A)TRANSFERASE?
 L2 1105 S "GST-ALPHA" OR GST4(W)ALPHA
 L3 3819 S L1 OR L2
 L4 1242 S HUMAN AND L3
 L5 50 S HUMAN(W)L3
 L6 83 S HUMAN(2W)L3
 L7 6615667 S CLON? OR EXPRESS? OR RECOMBINANT
 L8 1242 S L4 OR L5 OR L6
 L9 700 S L7 AND L8
 L10 14569 S L(W)SELECTIN OR "L-SELECTIN"
 L11 0 S L9 AND L10
 L12 55768 S SELECTIN
 L13 3 S L9 AND L12
 L14 3 DUP REM L13 (0 DUPLICATES REMOVED)
 L15 63891 S "GLYCAM-1" OR "CD34" OR "MADCAM-1" OR "SGP200" OR "PODOCALYXI
 L16 9 S L9 AND L15

L17 4 DUP REM L16 (5 DUPLICATES REMOVED)
L18 33 S L5 AND L7
L19 12 DUP REM L18 (21 DUPLICATES REMOVED)
E ROSEN S/AU
L20 2426 S E3
L21 0 S L3 AND L20
L22 23 S GLYCOSYL (2W)SULFOTRANSFERASE?
L23 0 S L20 AND L22
L24 6 S HUMAN (2W)L22
L25 6 DUP REM L24 (0 DUPLICATES REMOVED)

=>

	Issue Date	Pages	Document ID	Title
1	20030925	101	US 20030180321 A1	Mycobacterial sulfation pathway proteins and methods of use thereof
2	20030605	98	US 20030104001 A1	Mycobacterial sulfation pathway proteins and methods of use thereof
3	20021107	36	US 20020164748 A1	Glycosyl sulfotransferase-3
4	20011213	27	US 20010051370 A1	Glycosyl sulfotransferase-3
5	20020528	24	US 6395882 B1	Selectin ligands
6	20020430	18	US 6380371 B1	Endoglycan: a novel protein having selectin ligand and chemokine presentation activity
7	20020402	39	US 6365365 B1	Method of determining whether an agent modulates glycosyl sulfotransferase-3
8	20010724	27	US 6265192 B1	Glycosly sulfortransferase-3

	Issue Date	Pages	Document ID	Title
1	20040415	37	US 20040072290 A1	Glycosylation engineering of antibodies for improving antibody-dependent cellular cytotoxicity
2	20040415	42	US 20040071686 A1	Treatment of alpha-galactosidase A deficiency
3	20031204	794	US 20030224376 A1	Novel human transferase family members and uses thereof
4	20031120	30	US 20030215835 A1	Differentially-regulated prostate cancer genes
5	20030522	15	US 20030096366 A1	Method for production of recombinant proteins in eukaryote cells
6	20030515	61	US 20030092160 A1	Recombinant protein production in a human cell
7	20030410	34	US 20030068818 A1	Animal tissue with carbohydrate antigens compatible for human transplantation and a carbohydrate determinant selection system for homologous recombination
8	20021107	36	US 20020164748 A1	Glycosyl sulfotransferase-3
9	20021031	48	US 20020160979 A1	Methods for inhibiting angiogenesis
10	20021024	55	US 20020155499 A1	32624, a novel human UDP-glucuronosyl and glycosyl transferase family member and uses thereof

	Issue Date	Pages	Document ID	Title
11	20021017	14	US 20020151471 A1	Factor VII glycoforms
12	20020926	14	US 20020137673 A1	Factor VII glycoforms
13	20020822	85	US 20020115628 A1	47169 and 33935, novel human glycosyl transferases and uses thereof
14	20020620	11	US 20020076740 A1	PROCESS FOR GLUCURONIDATION SCREENING
15	20020328	69	US 20020037850 A1	Novel polypeptides and nucleic acids encoding same
16	20011213	27	US 20010051370 A1	Glycosyl sulfotransferase-3
17	20011213	201	US 20010051335 A1	POLYNUCLEOTIDES AND POLYPEPTIDES DERIVED FROM CORN TASSEL
18	20040302		US 6699654 B1	Antimicrobial agents diagnostic reagents, and vaccines based on unique apicomplexan parasite components
19	20040224	258	US 6696561 B1	Corynebacterium glutamicum genes encoding proteins involved in membrane synthesis and membrane transport
20	20030805		US 6602684 B1	Glycosylation engineering of antibodies for improving antibody-dependent cellular cytotoxicity
21	20030422		US 6551790 B2	Process for glucuronidation screening
22	20030415		US 6548643 B1	Antigen carbohydrate compounds and their use in immunotherapy

	Issue Date	Pages	Document ID	Title
23	20020402	39	US 6365365 B1	Method of determining whether an agent modulates glycosyl sulfotransferase-3
24	20011120		US 6319678 B1	Process for glucuronidation screening
25	20010731		US 6268484 B1	HIV-vaccines
26	20010724	27	US 6265192 B1	Glycosly sulfotransferase-3
27	20010123		US 6177256 B1	Antigen carbohydrate compounds and their use in immunotherapy
28	19991123		US 5989552 A	Antigen carbohydrate compounds and their use in immunotherapy
29	19990615		US 5911989 A	HIV-vaccines
30	19990216		US 5871950 A	Methods of treating autoimmune diseases and transplantation rejection

	Issue Date	Pages	Document ID	Title
31	19970930		US 5672692 A	Purification of human myelomonocyte interferon gamma with an immobilized antibody
32	19970715		US 5648218 A	Preparation of photoprotein conjugates and methods of use thereof
33	19960917		US 5556754 A	Methods for assessing the ability of a candidate drug to suppress MHC class I expression
34	19960910		US 5554515 A	Preparation of a monoclonal antibody specific to human myelomonocyte interferon-gamma
35	19960521		US 5518899 A	Preparation of human myelomonocyte interferon-gamma
36	19960123		US 5486455 A	Photoprotein conjugates and methods of use thereof
37	19941108		US 5362490 A	Human myelomonocyte interferon-gamma, and process for preparation and use thereof
38	19900717		US 4942131 A	Monoclonal antibody and method for preparation of hybridoma producing said antibody

	Issue Date	Pages	Document ID	Title
39	19870707		US 4678747 A	Monoclonal antibodies for detection of an H (O) blood group antigen

	Issue Date	Pages	Document ID	Title
1	20021107	36	US 20020164748 A1	Glycosyl sulfotransferase-3
2	20011213	27	US 20010051370 A1	Glycosyl sulfotransferase-3
3	20020402	39	US 6365365 B1	Method of determining whether an agent modulates glycosyl sulfotransferase-3
4	20010724	27	US 6265192 B1	Glycosly sulfortransferase-3

	Issue Date	Pages	Document ID	Title
1	20021107	36	US 20020164748 A1	Glycosyl sulfotransferase-3
2	20011213	27	US 20010051370 A1	Glycosyl sulfotransferase-3
3	20020402	39	US 6365365 B1	Method of determining whether an agent modulates glycosyl sulfotransferase-3
4	20010724	27	US 6265192 B1	Glycosly sulfortransferase-3
5	20000801	47	US 6096512 A	Cloned DNA encoding a UDP-GalNAc: Polypeptide, N-acetylgalactosaminylt ransferase
6	19990608	56	US 5910570 A	Cloned DNA encoding a UDP-GalNAc: polypeptide N-acetylgalactosaminy-l transferase
7	19870707	7	US 4678747 A	Monoclonal antibodies for detection of an H (O) blood group antigen

	L #	Hits	Search Text
1	L1	8	glycosyl adj sulfotransferase\$2
2	L2	42287 6	human
3	L3	8	11 and 12
4	L4	64356 1	clon\$3 or express\$3 or recombinant
5	L5	673	glycosyl adj3 transferase\$2
6	L6	80	human same 15
7	L7	39	14 same 16
8	L8	3913	selectin
9	L9	4	17 same 18
10	L10	19768	ROSEN
11	L11	7	16 and 110